Medical Staff Conference

Refer to: Complications of vomiting—The Boerhaave and the Mallory-Weiss syndromes—Medical Staff Conference, University of California, San Francisco. West J Med 121:50-54, Jul 1974

Complications of Vomiting

The Boerhaave and the Mallory-Weiss Syndromes

These discussions are selected from the weekly staff conferences in the Department of Medicine, University of California, San Francisco. Taken from transcriptions, they are prepared by Drs. David W. Martin, Jr., Assistant Professor of Medicine, and H. David Watts, Assistant Professor of Medicine, under the direction of Dr. Lloyd H. Smith, Jr., Professor of Medicine and Chairman of the Department of Medicine. Requests for reprints should be sent to the Department of Medicine, University of California, San Francisco, CA 94143.

DR. SMITH:* *Vomiting is basically a protective function which may even on occasion be life saving. As is the case with many such functions, however, it may carry with it its own complications ranging from the unpleasant to the dangerous. Dr. David Watts has recently reviewed a series of our patients with the Mallory-Weiss syndrome. This morning he will discuss this syndrome and other problems which may be associated with vomiting.*

DR. WATTS:† Vomiting is possibly one of the least pleasant experiences of life. If it has a positive side, it is that vomiting can on occasion relieve gastric distress and can clear the stomach of noxious or disagreeable substances. Though aesthetically somewhat wanting, it has over the ages commanded considerable attention as a therapeutic tool. Indeed, various concoctions, potions and natural spirits have been used to induce emesis in the hopes of relieving some of the abdominal woes of man. Galen, for example, recommended administering warm water and oil to patients with opium poisoning, until vomiting occurred.

Emetics commonly used in the United States at the turn of the century included some that were frank poisons themselves but were capable, fortunately, of inducing emesis before significant amounts were absorbed.

In short, vomiting has long been considered desirable under certain circumstances. It is now quite clear, however, that this violent assault upon the stomach and esophagus can result in damage to these structures, producing a wide range of lesions. These range from simple tears of the mucosa to, in the more severe circumstances, the very catastrophic, through-and-through ruptures. The Mallory-Weiss lesion is the most well known of emetogenic lesions, but it is inseparably linked with a number of other lesions that can result from retching or vomiting. These include the Boerhaave lesion, intramural hematomas, intramural perforation and gastric perforation.

Gastric Perforation

Gastric perforation is included here for the sake of completeness, though this lesion is not usually postemetic. Most cases of perforation of the stomach are due to intragastric disease processes, such as gastric ulcer or carcinoma, or are caused by trauma due to sharp penetrating missiles

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or severe blunt trauma to the abdomen. In a small number of patients, however, there may be a so-called spontaneous rupture of the stomach, mostly associated with vomiting or overeating or both.[1,2]

Intramural Esophageal Perforation

An intramural esophageal perforation is a lesion that occurs as a result of continued retching and vomiting which causes the mucosa to become lacerated and a small false lumen of the esophagus to be formed.[3,4] The lumen may dissect for some distance or remain relatively localized. This lesion basically can be regarded as an incomplete Boerhaave lesion, and may respond to conservative measures alone.[3]

Intramural Hematomas

Intramural hematomas were first described in the literature in 1965.[5] The most dramatic cases were reported later by Thompson, Ernst and Frye.[6] In one of their patients a dissecting hematoma encased the entire esophagus, and in another it dissected along the lesser curvature of the stomach. Both required surgical operation, and both could be traced to small mucosal lesions in the region of the cardio-esophageal junction.

A somewhat milder form of this lesion was recently reported under the name esophageal apoplexy.[7] Three patients were described, all elderly ladies with intramural hematomas occurring at the cardio-esophageal (C-E) junction. Despite the rather dramatic presentation of pain and hematemesis, and esophageal filling defect seen by x-ray studies and endoscopic examination, all three responded to conservative management.

The Boerhaave Lesion

The first description of an emetogenic lesion of the esophagus is attributed to the Dutch surgeon, Hermann Boerhaave. His report of the patient and the lesion was contained in a 70-page monograph which appeared two and one-half centuries ago this year.[8] His famous patient was the Grand Admiral of the Netherlands, Baron von Wassenauer.

It seems that the Baron was accustomed, as were others of his station, to great gastronomic overindulgence. This was sometimes followed by self-induced emesis when such practices caused him distress. He was in the habit of taking a copious draught of an infusion of "blessed thistle" and an ipecac-like substance. One evening in the late 1600's, the Baron became very distressed, for he downed three tumblersful of his favorite remedy and when it failed to work, gagged himself. He experienced sudden excruciating chest pain, saying he felt something had broken inside. He was right. When he died a short time later, Dr. Boerhaave performed an autopsy and found the esophageal perforation that now bears his name.

The Boerhaave lesion is a very predictable one. It occurs almost invariably in the distal esophagus, 1 to 3 cm above the C-E junction, is arranged longitudinally and located on the left posterolateral wall. This lesion has been easily reproduced in human cadaver esophagi tied off and blown up like a balloon until they burst.[9] However, recognizing that this was not the most physiologic of circumstances, Bodi et al[10] altered the experimental design by leaving the cadaver esophagus *in situ,* and *not* obstructing the upper esophagus. With a rapidly moving bolus of fluid injected through the cardia and out the esophagus the Boerhaave lesion could be reproduced, leading to the postulate that the sudden filling of the lower esophagus with a sizable volume of fluid caused rapid distension and rupture.

It was not until 1947 that the first successful surgical closure of a Boerhaave lesion was done.[11] Today, the increased success rate for this procedure depends on rapid diagnosis, wide debridement of necrotic esophageal and mediastinal tissue, adequate postoperative drainage and broad spectrum antibiotic coverage.

The Boerhaave lesion is regarded as the most rapidly fatal and most serious perforation of the gastrointestinal tract. An immediate chemical mediastinitis occurs, manifested by excruciating chest pain and hypotension, and may be easily mistaken for a myocardial infarction. The best route to correct diagnosis is a high index of suspicion and good x-ray studies of the chest. Mediastinal emphysema occurs early and can be auscultated over the pericardium. This sign is commonly known as Hammond's crunch. The emphysema can be seen on x-ray films in the mediastinum, or when it dissects far enough superiorly as it commonly does, it may be palpated subcutaneously above the clavicles. A left pneumothorax and pleural effusion occurs in approximately 80 percent of cases. Rupture through the pleura may occur, either immediately or later, as a result of the intense inflammatory response. Rupture into the pericardium has also been reported. The pleural fluid will show a significant

amylase activity, thus causing possible confusion with another diagnostic possibility, acute pancreatitis. The amylase in the pleural fluid of patients with Boerhaave syndrome migrates electrophoretically with salivary amylase, thus emphasizing the contribution of swallowed secretions to the genesis of the effusion.[12] Also of help in distinguishing it from pancreatitis is the fact that serum amylase activity is not elevated in patients with the Boerhaave lesion alone.

The prognosis of untreated patients is very grave. In a study by Derbes and Mitchell,[13] a careful review of survival in the era before surgery was conducted. Twenty-five percent of all patients studied died within 12 hours of the rupture, 65 percent within the first 24 hours and the remainder by the end of the week. This was at a time when good antibiotic coverage was not available, and although there are now scattered reports of nonsurgical cures, such cases still merit reporting. Hence, a misdiagnosis is almost invariably fatal. In the rare case where symptoms of mediastinitis are minimal, ingestion of water-soluble contrast media may help make the diagnosis.

Shock is not a contraindication to operation in these patients; rather, it underscores the necessity for surgical intervention, for upon opening the mediastinum and release of tamponade, blood pressure is often revived. Postoperatively, wound infection with resultant dehiscence or formation of bronchopleural fistula are the most feared complications. Therefore, adequate debridement during operation and placement of drains before closing are imperative. Despite these problems, reported survival rates range from 60 to 90 percent of cases in which operation is done.[13]

A Boerhaave lesion may on rare occasions bleed if the tear disrupts vessels of a significant size,[14] but it is not likely that confusion will arise in distinguishing it from a Mallory-Weiss lesion because of its associated signs and symptoms.

Having discussed an emetogenic lesion with rather clear surgical indications, we move to one about which there perhaps is still some controversy.

The Mallory-Weiss Lesion

The Mallory-Weiss lesion was first described in 1929. In their original paper, Mallory and Weiss[15] described 15 patients each of whom, after a long and intense alcohol debauch, developed massive gastric hemorrhage with hematemesis. During autopsy studies of four of these patients, between two and four fissure-like lesions of the mucosa of the gastric cardia were found in each. Characteristically, these lesions were arranged around the circumference of the cardiac opening along the longitudinal axis of the esophagus. Two factors were considered as causes: alcohol and vomiting. The investigators were aided in incriminating the latter by the occurrence of an identical lesion in a patient with pernicious vomiting in pregnancy.

Mallory and Weiss felt strongly that these lesions occurred only when disco-ordinate vomiting occurred. This might occur when repeated episodes of vomiting lead to "fatigue" of the vomiting center, resulting in forceful and prolonged contractions of the muscles of retching. Since we now know that this lesion may occur without repeated episodes of emesis preceding it, it is no longer necessary to rely upon this hypothesis.

A rather large number of the various causes leading to vomiting and producing the Mallory-Weiss lesion was listed by Weaver, Maxwell and Castleton in 1969.[16] Alcohol was a decided influence in 60 percent, but almost any imaginable cause for vomiting can result in the Mallory-Weiss tear, including several iatrogenic forms. It is also important to note that the lesion may be produced by factors other than vomiting that cause sudden increases in intra-abdominal pressure such as straining at stool or sudden heavy lifting, or even cardiac massage. A clear history of preceding episodes of vomiting is *not* always present. In fact, recent reports state that vomiting or retching before the occurrence of the lesion is only found in the history of some 50 percent of patients.[17,18] It appears, therefore, that in a sizable number of these patients the lesion may develop with the first episode of vomiting. This emphasizes the fact the Mallory-Weiss tear can certainly occur in the absence of a classic history.

Hiatus hernia has often been considered to be a predisposing condition to the development of the Mallory-Weiss lesion.[19] With sudden descent of the diaphragm and increased intra-abdominal pressure, intragastric pressure rises sharply. The contents of the stomach are forced upward, causing dilatation of the hiatus hernia (HH) pouch. The pouch is located in the thorax where there is less extraluminal pressure, and considerable ballooning of the pouch may occur and lead to tearing of the mucosa. The incidence of HH in the Mallory-Weiss syndrome is 50 percent, but since

a temporary hiatus hernia can be caused by retching in many "normals," this datum is not helpful. What this theory fails to explain, however, is how such lesions may occur in the distal esophagus, as around 30 percent of them do.[18] We are forced to conclude, I think, that the existing theory of the pathogenesis of this lesion is inadequate. An alternative explanation comes from the observation of retching during endoscopic examination. Depression of the diaphragm can be seen intralumenally as a constricting ring moving down a relatively stationary esophageal tube with some ballooning occurring proximally as the ring passes. Shearing forces generated by movement of esophageal mucosa through this ring may contribute to the genesis of such a lesion. Since the diaphragm commonly rests 1 to 2 cm above the C-E junction, distal esophageal tears can be explained by this hypothesis.

How does one arrive at the diagnosis of a Mallory-Weiss tear? The classic history of repeated retching will provide a clue in probably less than half of the cases. X-ray studies are not generally helpful. Sporadic cases of diagnosis from x-ray films have been reported. In these instances the radiologist was fortunate enough to catch contrast media in the Mallory-Weiss trough,[20,21] but this is the exception rather than the rule.

Endoscopy is by far the best diagnostic tool. From our experience with this disorder, recent lesions appear bloody and fresh. Those that are a few hours old will be covered by a large clot. By the third to fourth day the clot is gone, and the underlying scar tissue is apparent. There is complete healing by seven to eight days.

Standard treatment of this lesion includes lavage until clear, followed by administration of antacids. The use of tubes should be avoided. This is purely empiric therapy, and no controlled series exist to help evaluate it.

Use of a Sengstaken-Blackmore tube as a tamponading device against the bleeding site was advocated recently by Loren Pitcher,[22] but some observers have said that this might even extend the lesion.[23] It clearly adds another potentially complicating device to the regimen. For those patients in whom the bleeding does not stop, arteriography and vasopressin infusion has been used successfully by Dill and coworkers[24] to arrest hemorrhage.

It is our feeling, however, that the natural history of the lesion includes spontaneous arrest of hemorrhage in most cases and that the tip of the iceberg, obviously the most dramatic part, is what has been given the most attention.

With the advent of endoscopy the diagnosis of the Mallory-Weiss syndrome can be made with certainty. Widespread use of this technique has resulted in the appreciation of a higher frequency of diagnosis of this lesion (some 4.5 percent among all causes of significant upper gastrointestinal bleeding in some studies).[18,25] The fact that this lesion is benign in a significant number of patients has recently caught our attention and led to the observation that average blood loss is approximately two units (39 percent of 36 patients not requiring any transfusion at all), surgical intervention is necessary in 19 percent, and mortality was very low (3 percent).[18]

Thus, we have seen some changes in our conception of the Mallory-Weiss syndrome: (1) That a history of vomiting preceding hematemesis is not at all necessary; (2) that the lesion itself does not usually bleed massively; (3) the hemorrhage has a tendency to arrest spontaneously or in response to conservative medical treatment, and (4) that the mortality rate is quite low. It seems likely also that smaller, subclinical lesions commonly develop but escape detection simply because of the lack of significant symptoms. Hence, the lesion is probably a great deal more common than is realized. As a result of these observations and conclusions, we can say that conservative management is indicated until it becomes clearly apparent that medical therapeutic methods have been exhausted or until the patient's clinical course departs seriously from the natural history of the lesion.

A common remedy in the ancient writings of the Torah was a mixture of roots and herbs in beet juice which the patient was given to drink. Although it usually cured the patient, it unfortunately rendered him impotent. This example has some applicability to the Mallory-Weiss syndrome. In closing I submit that in medicine generally, and perhaps specifically in the case of the Mallory-Weiss lesion, the physician must make very certain that the cure he recommends is not inappropriate or perhaps even worse than the disease itself.

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Legal Implications of Laparoscopic Complications

LET ME JUST COVER THE HIGHLIGHTS of what might concern you in regard to laparoscopy. The informed consent doctrine applies differently in different states. Some states require more disclosures by physicians to patients than do other states in order to secure an adequately-informed legal consent. But let me try to give some minimal concept as to what probably would protect you in most states. If we can avoid considering laparoscopy as simple (and we can always avoid telling a patient it is simple) you have avoided 90 percent of the problem of informed consent. It's as simple as that. You never, never tell a patient that any laparoscopic procedure, either for diagnostic or therapeutic purposes, is simple; and you will have avoided most of the problems associated with it.

I think that all of your laparoscopic procedures should have some type of written consent form. Somewhere in the middle of the form, put the statement that this procedure, like all operative procedures, may be subject to serious complications or even death. You can use that language somewhere in the middle of your form. Now, I am not telling you to list all of the complications. I'm not telling you not to, either. But if that is the minimal disclosure required, I don't think it is going to harm many patients. First of all, they have to read it. Second of all, they have to understand it. And most patients realize the generalities of "serious complications or death." And as long as there is evidence of some type of disclosure on a minimal basis such as that, I think you've avoided another 9 percent of the problem, in addition to the 90 percent avoided by not telling them it's simple. If there is any question concerning the indications for the procedure, then you have to go farther. There's no way of justifying the performance of a non-indicated procedure without the patient's knowing it's not indicated . . . or without the patient's knowing that there are questions concerning its indications. If that's true, then you've got to disclose it and go further in terms of the risks involved, so that the patient is then making a decision along with you as to whether the procedure should be performed at all . . ."

—DON H. MILLS, MD, JD, *Los Angeles*

Extracted from *Audio-Digest Obstetrics and Gynecology*, Vol. 21, No. 3, in the Audio-Digest Foundation's subscription series of tape-recorded programs. For subscription information: 1930 Wilshire Blvd., Suite 700, Los Angeles, CA 90057.